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=> s antisense and phosphorothioate and glutathione
L1          25 ANTISENSE AND PHOSPHOROTHIOATE AND GLUTATHIONE

=> dup l1 remove
PROCESSING COMPLETED FOR L1
L2          12 DUP REMOVE L1 (13 DUPLICATES REMOVED)

=> s l2 and py=<2000
   1 FILES SEARCHED...
   3 FILES SEARCHED...
L3          11 L2 AND PY=<2000

=> d l3 bib abs 1-11

L3   ANSWER 1 OF 11  BIOSIS  COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN   2000:176756  BIOSIS
DN   PREV200000176756
TI   In vivo electroporetic transfer of bcl-2 antisense
     oligonucleotide inhibits the development of hepatocellular carcinoma in
     rats.
AU   Baba, Miyako (1); Iishi, Hiroyasu; Tatsuta, Masaharu
CS   (1) Department of Gastrointestinal Oncology, Osaka Medical Center for
     Cancer and Cardiovascular Diseases, 3-3, Nakamichi 1-chome,
     Higashinari-ku, Osaka, 537-0025 Japan
SO   International Journal of Cancer., (Jan. 15, 2000) Vol. 85, No.
     2, pp. 260-266.
     ISSN: 0020-7136.
DT   Article
LA   English
SL   English
AB   To investigate the potential use of a bcl-2 antisense
     oligonucleotide for therapy against hepatocellular carcinoma, we examined
     the effects of the electroporetic transfer of a bcl-2 antisense
     oligonucleotide on rat hepatocarcinogenesis induced by N-nitrosomorpholine
     (NNM). Sprague-Dawley rats were given water containing 175 mg/l NNM for 8
     weeks and received intraperitoneal injections of a bcl-2 antisense
     phosphorothioate oligonucleotide, a sense oligonucleotide or a
     scrambled sequence oligonucleotide encapsulated in empty liposomes, at a
     dose of 150 mug oligonucleotide/kg body weight, every 4 weeks. One hour
     after injection, in vivo electroporation was performed on the liver to
     achieve selective transfer of the oligonucleotides. By week 16, the rats
     that had received the bcl-2 antisense oligonucleotide had
     significantly fewer and smaller precancerous liver lesions positive for
     glutathione-S-transferase (placental type), and a significantly
     lower incidence of hepatocellular carcinoma in the electroporation zone
     than rats that had received the sense or the scrambled oligonucleotides.
     Moreover, the bcl-2 anti-sense oligonucleotide significantly increased the
     apoptotic indices in foci, neoplastic nodules and in hepatocellular
     carcinomas. The expression of bcl-2 mRNA also decreased, and 3'-fragments
     of bcl-2 mRNA produced by cleavage at the antisense target site
     were detected in rat liver. Mean cellular fluorescence in the liver
     increased with higher doses of fluorescein-isothiocyanate-labeled
     antisense or sense oligonucleotides. Our results show that the
     electroporetic transfer of bcl-2 antisense oligonucleotide can
     inhibit rat hepatocarcinogenesis.

L3   ANSWER 2 OF 11  BIOSIS  COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN   1997:456974  BIOSIS
DN   PREV199799756177
TI   Ha-ras mutations in N-nitrosomorpholine-induced lesions and inhibition of
     hepatocarcinogenesis by antisense sequences in rat liver.
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AU Baba, Miyako (1); Yamamoto, Reiko; Iishi, Hiroyasu; Tatsuta, Masaharu
CS (1) Dep. Gastrointestinal Oncol., Osaka Med. Cent. Cancer, Cardiovascular Diseases, 3-3 Nakamichi 1-chome, Higashinari-ku, Osaka 537 Japan
SO International Journal of Cancer, (1997) Vol. 72, No. 5, pp. 815-820. ISSN: 0020-7136.
DT Article
LA English
AB To evaluate the application of Ha-ras mRNA **antisense** oligonucleotide therapy for liver tumors, we examined the frequency and types of mutation in codon 61 of the Ha-ras oncogene in preneoplastic lesions and hepatocellular carcinomas induced by N-nitrosomorpholine (NNM) in rats. Thirty-seven percent of preneoplastic lesions and 50% of hepatocellular carcinomas contained mutations, mostly CAA-CTA and CAA-AAA transversions. We also investigated the effects on NNM-induced lesions of an **antisense** oligonucleotide directed against a point mutation (CAA-CTA) in codon 61 of Ha-ras mRNA. In this experiment, Sprague-Dawley rats were given free access to water containing NNM for 8 weeks and received twice-weekly i.p. injections of a mutated Ha-ras **antisense** oligonucleotide with a 5' **phosphorothioate** linkage or a sense oligonucleotide in oligonucleotide-liposome complexes. At week 16, rats that had received the mutated Ha-ras **antisense** oligonucleotides had significantly fewer and smaller preneoplastic lesions positive for **glutathione**-S-transferase, placental type, and had smaller hepatocellular carcinomas than rats that had received the sense oligonucleotide. Mean cellular fluorescence in the liver was found to increase with higher doses of mutated, fluorescein-isothiocyanate-labeled **antisense** or sense oligonucleotides. Moreover, mutated Ha-ras **antisense** oligonucleotide decreased the expression of mutated Ha-ras mRNA (CAA-CTA). Our findings indicate that mutated Ha-ras **antisense** oligonucleotide significantly inhibits hepatocarcinogenesis in rats and could be an effective therapy against liver tumors.

L3 ANSWER 3 OF 11 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1995:345198 BIOSIS
DN PREV199598359498
TI **Phosphorothioate** oligonucleotides bind in a non sequence-specific manner to the nucleolar protein C23/nucleolin.
AU Weidner, Douglas A.; Valdez, Benigno C.; Henning, Dale; Greenberg, Scott; Busch, Harris (1)
CS (1) Dep. Pharmacology, Baylor Coll. Med., Houston, TX 77030 USA
SO FEBS Letters, (1995) Vol. 366, No. 2-3, pp. 146-150. ISSN: 0014-5793.
DT Article
LA English
AB To design optimal strategies for intracellular delivery of **antisense phosphorothioate** oligonucleotides, it may be useful to understand their interaction with cellular macromolecules. Nuclear extracts from LOX amelanotic myeloma cells were studied for protein binding to **phosphorothioate** oligonucleotides using a Southwestern protocol. Multiple nuclear proteins bound to the **phosphorothioate** oligonucleotides but no detectable protein binding was found to phosphodiester oligonucleotides. The protein with the strongest binding signals was shown by immunoprecipitation to be nucleolar C23/nucleolin, a 110 kDa protein. With **glutathione** S-transferase/nucleolin fusion protein constructs, the region of nucleolin containing the RNA recognition motifs had binding activity to **phosphorothioate** oligonucleotides.

L3 ANSWER 4 OF 11 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1995:340622 BIOSIS
DN PREV199598354922

TI Coordinate Expression and Developmental role of Id2 Protein and TAL1/E2A Heterodimer in Erythroid Progenitor Differentiation.
AU Condorelli, Gianluigi; Vitelli, Luigi; Valtieri, Mauro; Marta, Isabella; Montesoro, Elisabetta; Lulli, Valentina; Baer, Richard; Peschle, Cesare (1)
CS (1) Thomas Jefferson Cancer Inst., Thomas Jefferson Univ., Bluemle Life Sci. Bldg. Rm. 528, 233 S. 10th St., Philadelphia, PA 19107-5541 USA
SO Blood, (1995) Vol. 86, No. 1, pp. 164-175.
ISSN: 0006-4971.
DT Article
LA English
AB The Id proteins and basic helix-loop-helix (bHLH) proteins play major roles in specifying cell fate decisions in diverse biologic settings. A potential role for Id and TAL1/E2A bHLH genes in hematopoiesis has been suggested by studies on immortalized cell lines. However, it is uncertain whether these observations reflect normal hematopoiesis. We have investigated the expression pattern of Id2 and TAL1/E2A genes in liquid suspension culture of purified hematopoietic progenitor cell (HPCs) undergoing erythroid or granulopoietic differentiation in the first culture week and maturation to terminal cells in the second week. In quiescent, freshly purified HPCs, Id2 mRNA is detected by reverse transcriptase-polymerase chain reaction (RT-PCR), whereas TAL1 and E2A mRNAs are not. At the onset of erythroid differentiation, Id2 mRNA is downregulated, while E2A and TAL1 mRNAs are concomitantly upregulated: their expression is further increased at erythroblast level. Conversely, Id2 is not downmodulated in granulopoietic culture, except for a late decline at day 10 to 12, while TAL1 and E2A are only transiently induced in the first week of granulopoietic differentiation. The expression pattern of the TAL1/E2A heterodimer, as evaluated by mobility shift assay, is consistent with RT-PCR results (except for lower levels of the heterodimer in late erythroid maturation). TAL1 protein level, analyzed by Western blot, shows a pattern consistent with gel-shift results. Functional experiments were performed an purified HPCs treated with **phosphorothioate antisense** oligodeoxynucleotides to Id2 or TAL1 mRNA. The results are strictly consistent with the expression studies: anti-Id2 oligomer (alpha-Id2) causes a significant dose-dependent increase of erythroid colony formation, whereas alpha-TAL1 induces a selective dose-related inhibitory effect on erythroid colonies, as compared with untreated or scrambled oligomer-treated control HPCs. Finally, murine and human **glutathione**-S-transferase (GST)-Id2 polypeptides compete the TAL1/E2A-specific DNA binding activity when added to the nuclear extracts derived from erythroid culture cells, thus indicating biochemical and suggesting functional interaction of Id2 with the TAL1/E2A complex. These novel observations indicate a coordinate expression and function of an inhibitory Id protein (Id2) and a stimulatory bHLH/bHLH heterodimer (TAL1/E2A) in normal erythroid differentiation.

L3 ANSWER 5 OF 11 SCISEARCH COPYRIGHT 2002 ISI (R)
AN 2001:30585 SCISEARCH
GA The Genuine Article (R) Number: 387ZV
TI Multidrug resistance-associated protein - reduction of expression in human leukaemia cells by **antisense phosphorothioate** olignucleotides
AU Niewiarowski W; Gendaszewska E; Rebowski G (Reprint); Wojcik M; Mikolajczyk B; Goss W; Soszynski M; Bartosz G
CS Polish Acad Sci, Ctr Mol & Macromol Studies, Dept Bioorgan Chem, H Sienkiewicza 112, PL-90368 Lodz, Poland (Reprint); Polish Acad Sci, Ctr Mol & Macromol Studies, Dept Bioorgan Chem, PL-90368 Lodz, Poland; Univ Lodz, Dept Mol Biophys, PL-90131 Lodz, Poland
CYA Poland
SO ACTA BIOCHIMICA POLONICA, (**JAN 2000**) Vol. 47, No. 4, pp.

1183-1188.
Publisher: ACTA BIOCHIMICA POLONICA, PASTEURA 3, 02-093 WARSAW, POLAND.
ISSN: 0001-527X.
DT Article; Journal
LA English
REC Reference Count: 20
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Multidrug resistance-associated protein (MRP1) causes cellular drug resistance in several cancer cell lines. In this paper we show that **antisense** oligonucleotides decrease MRP1 expression in human leukaemia cells. We investigated biological activity of a series of 12 linear **phosphorothioate** oligonucleotides, complementary to several regions of MRP1 mRNA. The oligonucleotides were administered to leukaemia HL60/ADR cells overexpressing MRP1 protein. Then, the level of MRP1 mRNA was determined by means of semiquantitative RT-PCR and the protein level by reaction with specific monoclonal antibodies. Some of the investigated **antisense** oligonucleotides decrease the expression level of the MRP1 protein by 46% and its mRNA level by 76%.

L3 ANSWER 6 OF 11 SCISEARCH COPYRIGHT 2002 ISI (R)
AN 1999:150101 SCISEARCH
GA The Genuine Article (R) Number: 166MX
TI Cell-surface protein disulfide isomerase catalyzes transnitrosation and regulates intracellular transfer of nitric oxide
AU Zai A; Rudd M A; Scribner A W; Loscalzo J (Reprint)
CS BOSTON UNIV, SCH MED, WHITAKER CARDIOVASC INST, CTR ADV BIOMED RES, MED CTR, EVANS DEPT MED, BOSTON, MA 02118 (Reprint); BOSTON UNIV, SCH MED, WHITAKER CARDIOVASC INST, CTR ADV BIOMED RES, MED CTR, EVANS DEPT MED, BOSTON, MA 02118
CYA USA
SO JOURNAL OF CLINICAL INVESTIGATION, (**FEB 1999**) Vol. 103, No. 3, pp. 393-399.
Publisher: ROCKEFELLER UNIV PRESS, 1114 FIRST AVE, 4TH FL, NEW YORK, NY 10021.
ISSN: 0021-9738.
DT Article; Journal
FS LIFE
LA English
REC Reference Count: 42
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Since thiols can undergo nitrosation and the cell membrane is rich in thiol-containing proteins, we considered the possibility that membrane surface thiols may regulate cellular entry of NO. Recently, protein disulfide isomerase (PDI), a protein that catalyzes thio-disulfide exchange reactions, has been found on the cell-surface membrane. We hypothesized that cell-surface PDI reacts with NO, catalyzes S-nitrosation reactions, and facilitates NO transfer from the extracellular to intracellular compartment. We observed that PDI catalyzes the S-nitrosothiol-dependent oxidation of the heme group of myoglobin (15-fold increase in the rate of oxidation compared with control), and that NO reduces the activity of PDI by 73.1 +/- 21.8% ($P < 0.005$). To assess the role of PDI in the cellular action of NO, we inhibited human erythroleukemia (HEL) cell-surface PDI expression using an **antisense phosphorothioate** oligodeoxynucleotide directed against PDI mRNA. This oligodeoxynucleotide decreased cell-surface PDI content by 74.1 +/- 9.3% and PDI folding activity by 46.6 +/- 3.5% compared with untreated or ''scrambled'' **phosphorothioate** oligodeoxynucleotide-treated cells ($P < 0.0001$). This decrease in cell-surface PDI was associated with a significant decrease in cyclic guanosine monophosphate (cGMP) generation after S-nitrosothiol exposure (65.4 +/- 26.7% reduction compared with control; $P < 0.05$), with no effect on cyclic adenosine monophosphate (cAMP) generation after prostaglandin

E-1 exposure. These data demonstrate that the cellular entry of NO involves a transnitrosation mechanism catalyzed by cell-surface PDI. These observations suggest a unique mechanism by which extracellular NO gains access to the intracellular environment.

L3 ANSWER 7 OF 11 SCISEARCH COPYRIGHT 2002 ISI (R)
AN 1998:499019 SCISEARCH
GA The Genuine Article (R) Number: ZV735
TI Translational inhibition of messenger RNA of the human pi class **glutathione** S-transferase by **antisense** oligodeoxyribonucleotide
AU Keller C; AliOsman F (Reprint)
CS UNIV TEXAS, MD ANDERSON CANC CTR, DEPT EXPT PEDIAT, SECT MOL THERAPEUT, BOX 169, HOUSTON, TX 77030 (Reprint); UNIV TEXAS, MD ANDERSON CANC CTR, DEPT EXPT PEDIAT, SECT MOL THERAPEUT, HOUSTON, TX 77030
CYA USA
SO CHEMICO-BIOLOGICAL INTERACTIONS, (**24 APR 1998**) Vol. 112, pp. 307-323.
Publisher: ELSEVIER SCI IRELAND LTD, CUSTOMER RELATIONS MANAGER, BAY 15, SHANNON INDUSTRIAL ESTATE CO, CLARE, IRELAND.
ISSN: 0009-2797.
DT Article; Journal
FS LIFE
LA English
REC Reference Count: 38
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB In this study, a T7 plasmid expression vector containing the cDNA of a variant human GST-pi gene, hGSTP1*C, was used to examine the translational inhibition of the GST-pi mRNA with **antisense** deoxyribonucleotides (AS-ONs), and to investigate the dependency of the inhibition on ribonuclease (RNAse) H, AS-ON and target mRNA sequence specificity and AS-ON backbone modification. Translational inhibition of hGSTP1*C mRNA showed significant AS-ON concentration-dependency and was both target mRNA and AS-ON sequence specific. Fully modified phosphoromonothioate AS-ONs were less inhibitory than their partial phosphoromonothioate analogs; unmodified AS-ONs were inactive. RNAse H enhanced the translational inhibition by AS-ON specific to the translation initiation region mRNA, and was associated with cleavage of the target mRNA at the site of AS-ON:mRNA hybridization. AS-ONs directed to the A --> G and C --> T transitions, unique to hGSTP1*C, were more RNAse H-dependent than AS-ONs directed against the translation initiation site, indicating a greater involvement of RNAse H-dependent mRNA cleavage in the mechanism of translational inhibition by AS-ON at the polymorphic site. These data suggest that AS-ONs provide a potentially effective means of specific down-regulation of the human GST-pi gene, and demonstrate that the sites of GST-pi gene allelo-polymorphism can be targeted to translationally down-regulate the different GST-pi gene variants, specifically and differentially targeted.

L3 ANSWER 8 OF 11 SCISEARCH COPYRIGHT 2002 ISI (R)
AN 97:674733 SCISEARCH
GA The Genuine Article (R) Number: XU704
TI Induction of the differentiation of HL-60 promyelocytic leukemia cells by vitamin E and other antioxidants in combination with low levels of vitamin D-3: possible relationship to NF-kappa B
AU Sokoloski J A; Hodnick W F; Mayne S T; Cinquina C; Kim C S; Sartorelli A C (Reprint)
CS YALE UNIV, SCH MED, DEPT PHARMACOL, 333 CEDAR ST, NEW HAVEN, CT 06520 (Reprint); YALE UNIV, SCH MED, DEPT PHARMACOL, NEW HAVEN, CT 06520; YALE UNIV, SCH MED, DEPT EPIDEMIOL & PUBL HLTH, NEW HAVEN, CT 06520; YALE UNIV, SCH MED, CTR CANC, DEV THERAPEUT PROGRAM, NEW HAVEN, CT 06520

CYA USA
SO LEUKEMIA, (**SEP 1997**) Vol. 11, No. 9, pp. 1546-1553.
Publisher: STOCKTON PRESS, HOUNDMILLS, BASINGSTOKE, HAMPSHIRE, ENGLAND RG21 6XS.
ISSN: 0887-6924.
DT Article; Journal
FS LIFE
LA English
REC Reference Count: 39
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Epidemiological studies have provided evidence that diets rich in antioxidant nutrients may reduce the risk of cancer. To evaluate the possibility that dietary phytochemicals with antioxidant potential would create an environment capable of affecting the differentiation of HL-60 leukemia cells, we measured the effects of vitamin E and other dietary antioxidants on the differentiation produced by low levels of vitamin D-3 and analogs thereof. Vitamin E succinate and other antioxidant compounds (ie butylated hydroxyanisole, beta-carotene and lipoic acid) used alone had no significant effect on the differentiation of HL-60 cells; however, these agents markedly increased the differentiation produced by vitamin D-3. Previous studies from this laboratory have shown that a sequence-specific **antisense phosphorothioate** oligonucleotide to the Rel A subunit of NF-kappa B enhanced the differentiation of HL-60 cells produced by several inducing agents. Consistent with these observations, vitamin E succinate caused a marked reduction in the nuclear content of NF-kappa B bath in the presence and absence of vitamin D-3. These findings suggest that MF-kappa B may be a factor in regulating the differentiation of myeloid leukemia cells. The results also indicate that combinations of vitamin D-3 and analogs thereof with dietary antioxidants may be useful in overcoming the differentiation block present in acute promyelocytic leukemia cells.

L3 ANSWER 9 OF 11 SCISEARCH COPYRIGHT 2002 ISI (R)
AN 95:486820 SCISEARCH
GA The Genuine Article (R) Number: RH954
TI REACTION BETWEEN METABOLICALLY ACTIVATED ACETAMINOPHEN AND **PHOSPHOROTHIOATE** OLIGONUCLEOTIDES
AU COPPLE B L (Reprint); GMEINER W M; IVERSEN P L
CS UNIV NEBRASKA, MED CTR, DEPT PHARMACOL, 600 S 42ND ST, OMAHA, NE, 68198 (Reprint); UNIV NEBRASKA, MED CTR, DEPT PHARMACEUT SCI, OMAHA, NE, 68198; UNIV NEBRASKA, MED CTR, EPPLEY INST RES CANC, OMAHA, NE, 68198
CYA USA
SO TOXICOLOGY AND APPLIED PHARMACOLOGY, (**JUL 1995**) Vol. 133, No. 1, pp. 53-63.
ISSN: 0041-008X.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 34
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Assessment of toxic or mutagenic risks associated with **phosphorothioate** oligonucleotides (PTO) is important. In vitro and in vivo data have shown that PTOs are nontoxic and nonmutagenic. However, these studies do not address interactions between PTOs and other compounds. The sulfur on PTOs may provide a novel reactive center on a DNA molecule for drug interactions. This study chose acetaminophen (ACAP) as a model drug because ACAP is oxidized to the reactive intermediate N-acetyl-p-benzoquinone imine (NAPQI), which reacts with sulfur-containing compounds. Reaction of dCTP(S) with NAPQI or activated ACAP formed a product distinct from the reactants. Analysis of the product by fast atom bombardment mass spectroscopy gave a molecular weight consistent with NAPQI bound to a sulfur. Higher-molecular-weight products were seen on a

polyacrylamide gel electrophoresis after incubation of fluorescein-labeled PTO with NAPQI. These products were not seen after incubation of a phosphodiester oligonucleotide with NAPQI. P-31 NMR analysis confirmed the existence of a heterogenous mixture of adducts between a PTO and NAPQI. Non-sequence-specific PTOs of various lengths were tested for their ability to reduce ACAP toxicity. Cell viability showed that larger PTOs provided greater protection. We evaluated the ability of NAPQI to cause mutations in the LacZ gene of pBluescript plasmid which contained **phosphorothioate** linkages at designed locations within the gene. In addition, the ability of ACAP to cause mutations in the HGPRT locus in cells grown in dATP(S)-containing medium was measured. No mutations were seen in either assay. Based upon these results, activated ACAP is reactive with PTOs in vitro, although this interaction is nontoxic and nonmutagenic. (C) 1995 Academic Press, Inc.

L3 ANSWER 10 OF 11 SCISEARCH COPYRIGHT 2002 ISI (R)
AN 94:753402 SCISEARCH
GA The Genuine Article (R) Number: PT755
TI METALLOTHIONEIN IN CARCINOGENESIS AND CANCER-CHEMOTHERAPY
AU EBADI M (Reprint); IVERSEN P L
CS UNIV NEBRASKA, MED CTR, DEPT PHARMACOL, 600 S 42ND ST, OMAHA, NE, 68198 (Reprint); UNIV NEBRASKA, MED CTR, EPPLEY INST RES CANC & ALLIED DIS, OMAHA, NE, 00000
CYA USA
SO GENERAL PHARMACOLOGY, (**NOV 1994**) Vol. 25, No. 7, pp. 1297-1310. ISSN: 0306-3623.
DT General Review; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 125
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB 1. Despite considerable progress, cancer continues to remain the number one health threat to human beings. Currently, the targeted antineoplastic therapy is based on an understanding of the molecular mechanisms that govern the normal proliferation and functioning of the cellular elements. Furthermore, the gene-directed therapies and antibody-based approaches are also based on modulating specific signalling processes influencing growth factors and oncogenes that alter cellular proliferation.

2. The intracellular level of metallothionein, a low molecular weight metal binding protein consisting of 25-30% cysteine, containing no aromatic amino acids or disulfide bonds and binding between 5 and 7 g atoms of group II B heavy metals per mole protein, may play an important role in regulating cellular responsiveness to DNA interactive antineoplastic agents. For example, cells with acquired resistance to cisplatin or chlorambucil overexpress metallothionein, which tends to bind these alkylating agents to a higher extent than the non-resistant cells. Since humans synthesize several isoforms of metallothionein. It is not certain which isoforms are increased in cells with acquired resistance to anti-cancer drugs. In addition to sequestering electrophilic anti-cancer drugs, metallothionein, by regulating the activities of zinc-requiring metalloenzymes or scavenging radical species, may after the therapeutic efficacy of antineoplastic agents.

L3 ANSWER 11 OF 11 CA COPYRIGHT 2002 ACS
AN 136:112634 CA
TI Suppression of nuclear factor-.kappa.B-dependent processes using oligonucleotides
IN Nerenberg, Michael I.; Kitajima, Isao
PA Scripps Research Institute, USA
SO U.S. Pat. Appl. Publ., 18 pp., Cont.-in-part of U.S. Ser. No. 887,331, abandoned.
CODEN: USXXCO

DT Patent
LA English
FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
PI	US 2002006912	A1	20020117	US 1993-110161	19930820	
	WO 9535032	A1	19951228	WO 1994-US9350	19940819	<--
	W: AU, CA, FI, JP, NO, US, US					
	RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE					
	AU 9476711	A1	19960115	AU 1994-76711	19940819	<--
PRAI	US 1992-887331	B2	19920522			
	US 1993-110161	A	19930820			
	WO 1994-US9350	W	19940819			

AB **Antisense** oligonucleotides which hybridize with nuclear factor-.kappa.B (NF-.kappa.B) mRNA and methods of using these therapeutic oligonucleotides are disclosed. The invention relates to treatments for leukemia and septic shock.

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FULL ESTIMATED COST	46.57	46.78

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